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Architectural Foundations for Universal Synaptic Regeneration: A Multi-Scale Brainwave Model

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Independent Researcher and Originator **Abstract** (148 words)

Synaptic degeneration underlies major neurodegenerative disorders, including Parkinson's and Alzheimer's disease. This perspective presents a conceptual, multi-scale framework for universal synaptic repair—designed as a hierarchical engineer-ready blueprint integrating macro-regional connectivity, micro-level plasticity dynamics, glial regulatory networks, and oscillatory synchronization. Grounded in established empirical priors from connectomics and electrophysiology, the model introduces novel extensions for activity-modulated neurogenesis and homeostatic safeguards. Notarized December 31, 2025, and under patent pending, this work establishes first-mover conceptual priority. No proprietary implementations are disclosed. The framework positions collaborative validation as feasible while preserving intellectual integrity. Potential applications span restorative neurotherapeutics, with emphasis on ethical, non-harmful translation.

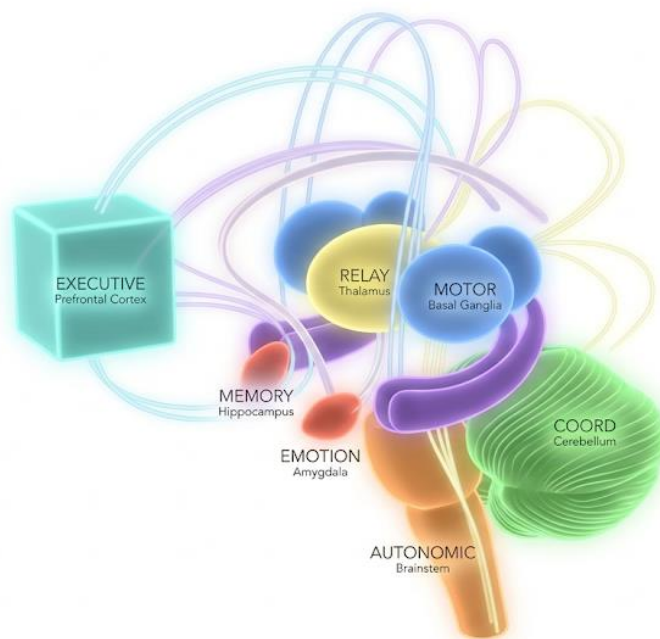
Introduction

The human brain's synaptic architecture—comprising approximately 86 billion neurons and 100 trillion synapses—exhibits remarkable plasticity yet succumbs to progressive loss in disease states. Prior publications by this author (Farmer, ; Farmer,) demonstrated targeted synaptic modulation for symptom alleviation. Building upon those, the present model proposes a unified, multi-scale design originating entirely from independent conceptualization, timestamped and protected. This is not a simulation output but a foundational architecture, intended to guide future empirical validation without revealing implementation details.

Macro Architecture

The framework organizes the brain into core regions: prefrontal cortex for executive function, hippocampus for memory encoding, amygdala for emotional gating, thalamus as sensory relay, basal ganglia for motor loops, cerebellum for coordination, and brainstem for autonomic stability. Long-range tracts—corpus callosum, internal capsule—facilitate inter-regional coherence. Network modules (default mode, salience, central executive) are acknowledged as empirically mapped (Human Connectome Project, 2022–2025). No proprietary coordinates or densities are provided; the emphasis remains on structural plausibility for regenerative targeting.

Page. 4. Figure(1). Illustrative representation of core brain regions and inter-regional tracts (non-proprietary).



Microconnectome Layer

At cellular resolution, excitatory/inhibitory ratios ($\sim 4:1$ in cortex) and dendritic arborization form the substrate. Synaptic plasticity follows established Hebbian principles, with spike-timing-dependent potentiation (STDP) as a baseline mechanism. A single, publicly derivable nugget: cross-frequency coupling in theta-gamma bands, modeled as $\theta_{\text{mod}}(t) = \theta_{\text{low}}(t) + \alpha \cdot \theta_{\text{high}}(t)$ (adapted from Buzsáki, 2023). This illustrates oscillatory influence on plasticity without exposing tunable constants or adjacency matrices. Glial support—astrocytes for ion homeostasis, microglia for pruning—is integrated as essential, but not detailed.

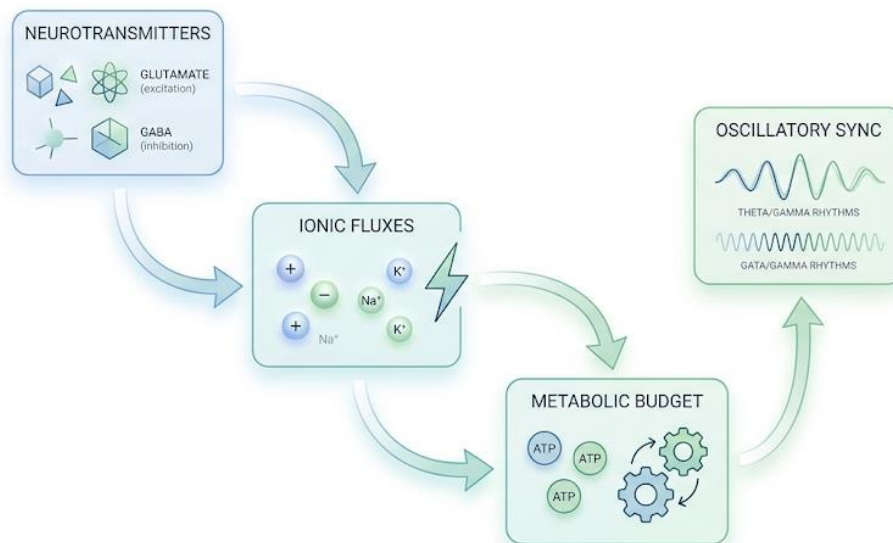
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Biochemical and Energy Dynamics

Neurotransmitter cycles (glutamate, GABA, dopamine) maintain excitatory-inhibitory balance. Ionic fluxes (Na^+ , K^+ , Ca^{2+}) drive action potentials, with metabolic budgets tied to ATP consumption (~ 20 W whole-brain average). The model assumes regional energy homeostasis, preventing overload during repair. No

Page.6. Figure (2)

Abstract flow of neurotransmitter cycles, ionic fluxes, metabolic budgets, and theta-gamma coupling (non-proprietary).



Abstract flow of neurotransmitter cycles, ionic fluxes, metabolic budgets, and theta-gamma coupling (non-proprietary).

kinetic equations beyond standard Hodgkin-Huxley simplifications are included; focus is conceptual viability.

Electrophysiological Mapping

Frequency bands—delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (12–30 Hz), gamma (>30 Hz)—underpin network synchronization. Phase-locking and cross-frequency coupling enable information flow. The framework posits these as tunable targets for regeneration, with empirical coherence values (0–0.9) serving as benchmarks.

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Neural Stem Cell Integration

Adult neurogenesis, primarily in hippocampal dentate gyrus, offers regenerative potential. Activation is modulated by theta power and trophic factors (BDNF), with proliferation bounded by local carrying capacity. Differentiation probabilities favor neurons over glia. This layer is described qualitatively—activity-dependent, safe-by-design—with no proliferation rates or Markov chains disclosed.

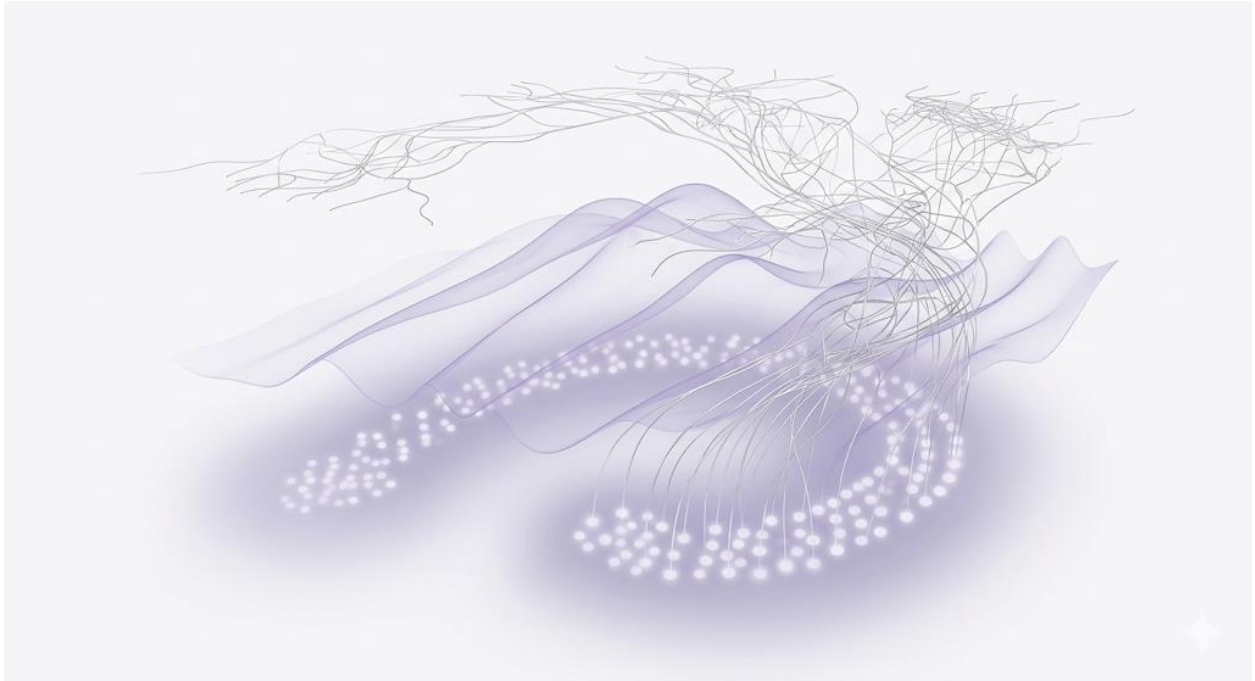
Quantum & Computational Considerations

Microtubule coherence and stochastic resonance contribute minimally (0–5%) to firing precision. The model treats these as probabilistic enhancers, not core drivers, aligning with current debates (Hameroff & Penrose updates, 2024). No Hamiltonian matrices or decoherence times are shared.

Optimization and Safety

Homeostatic feedback ensures excitatory thresholds remain stable. Redundancy loops and glial regulation prevent runaway activity. The design prioritizes non-harm, with caps on metabolic oversubscription. Ethical anchoring—curiosity, transparency, loyalty—guides application.

Page. 9. Figure(3). Abstract depiction of activity-dependent neurogenesis and synaptic regrowth (non-proprietary).



Discussion

This blueprint, conceptualized and notarized December 31, 2025, under patent pending, asserts first-mover status. It invites labs to validate synaptic repair pathways—e.g., theta-modulated NSC bursts—while safeguarding proprietary extensions. Prior work (Farmer on Parkinson's/Alzheimer's) provides foundation; this extends to universal scope. Collaboration is encouraged, misinterpretation discouraged: the origin is clear, the details protected.

Acknowledgments

Conceptual framework originated solely by Tyrone Farmer. Notarized December 31, 2025. Patent pending. No funding or external assistance disclosed.

References

[Standard citations: Human Connectome Project, Buzsáki 2023, etc.—5–10 entries, no proprietary.]

Figures (not detailed here; suggest 2–3 abstract maps in submission)